Welcome to Yale Cancer Answers with your host doctor in Anees Chagpar. Yale Cancer Answers features the latest information on cancer care by welcoming oncologists and specialists who are on the forefront of the battle to fight cancer. This week, it’s a conversation about using chemical tools to detect cancer causing proteins with doctor Stavroula Hatzios. Dr Hatzios is assistant professor of molecular,
cellular and developmental biology and of chemistry at the Yale School of Medicine where Doctor Chagpar is a professor of Surgical oncology.

Maybe we can start off by you telling us a little bit more about yourself and what it is you do. My background is in chemistry and microbiology so not a traditional cancer biologist, but I use chemical tools to better understand infectious diseases and more specifically how microbes can contribute to cancer in humans. So in my training I started out by
00:01:06.352 –> 00:01:07.870 researching infectious diseases,
00:01:07.870 –> 00:01:09.895 particularly airborne pathogens.
00:01:09.895 –> 00:01:11.920 Like Mycobacterium tuberculosis
00:01:11.920 –> 00:01:14.620 that causes human tuberculosis,
00:01:14.620 –> 00:01:15.840 but then as a postdoc,
00:01:15.840 –> 00:01:18.012 I switched to studying
00:01:18.012 –> 00:01:19.098 gastrointestinal pathogens,
00:01:19.100 –> 00:01:20.156 principally vibrio cholera,
00:01:20.156 –> 00:01:22.268 which is the bacterium that causes
00:01:22.268 –> 00:01:24.070 the diarrheal disease cholera.
00:01:24.070 –> 00:01:25.846 And it was through my postdoctoral
00:01:25.846 –> 00:01:28.184 training that I began to engage in
00:01:28.184 –> 00:01:29.644 conversations with other scientists
00:01:29.644 –> 00:01:31.719 who recommended that I start applying
00:01:31.719 –> 00:01:33.561 some of the chemical tools and
approaches that I was a developing
to study comparatively understudied microbes like Helicobacter pylori.
And that was really my entry point into the field of cancer microbiology and cancer microbiology really refers to an area of research that’s emerging where we’re looking at how microbes that indigenous microbes in our bodies. Which comprise the microbiome, as well as infectious microbes that cause disease might contribute to the development of cancer in humans or alter outcomes of cancer therapies. So I begin researching Helicobacter pylori a little bit as a postdoc,
and that’s really been the focal point of my labs work. Trying to understand how this very important gastric or stomach pathogen causes cancer in a subset of infected humans, and what are the pathways the molecular events by which cancer Develops and we use a lot of chemical approaches to sort of understand what those pathways are. Yeah, so I was going to ask Many people, when they think about Helicobacter pylori or H. Pylori, as it’s sometimes
00:02:41.034 –> 00:02:43.054 known we think about ulcers.

NOTE Confidence: 0.885287825833333

00:02:43.054 –> 00:02:45.430 We don’t really think about cancer.

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00:02:45.430 –> 00:02:47.446 So can you talk a little bit more about the link between H. Pylori and cancer and how how you got started with that?

NOTE Confidence: 0.885287825833333

00:02:49.390 –> 00:02:51.226 Absolutely yeah, H. Pylori is a fascinating, fascinating microbe.

NOTE Confidence: 0.842478474285714

00:02:55.694 –> 00:02:57.566 As you mentioned, it is primarily linked, at least in public knowledge, to peptic ulcers, stomach inflammation.

NOTE Confidence: 0.842478474285714

00:03:04.210 –> 00:03:07.410 But it’s also the leading risk factor for gastric cancer,

NOTE Confidence: 0.842478474285714

00:03:11.470 –> 00:03:13.480 which I think currently remains
the third leading cause of cancer worldwide. This is a microbe that’s found in half of the global population, and for most people it it doesn’t lead to cancer. It may actually be innocuous, meaning it may not do too much to the infected host, but a subset of those who carry the microbe as a normal part of their stomach microbiome will develop peptic ulcers and gastric inflammation, called gastritis. That’s roughly 10 to 15%
of people who have H. Pylori and then a much smaller percentage. 1 to 3% typically go on to develop gastric cancer and this connection was only made a couple of decades ago by Robin Warren and Barry Marshall, who won the Nobel Prize in medicine in 2005 for this discovery that this microbe can cause peptic ulcers, gastric inflammation and ultimately cancer and in fact due to their work, H. Pylori is now the first, formally characterized or classified microbe known to be a human carcinogen. So in individuals who have H.
the microbe can cause chronic inflammation of the gastric lining and overtime and some hosts. This can develop into gastric cancer. And what’s a major challenge for those of us in the field is trying to understand why it is that some individuals develop cancer and others do not. And that’s a really important question. The reason it’s so important is that typically if someone has H. Pylori, You can administer antibiotics to get rid of microbe, but these microbes have a
way of evolving very, rapidly and thus they evolve drug resistance, which limits the number of drugs available to treat them. So if you just administer antibiotics to half the global population to rid them of H. Pylori, that may not be the best approach because you’ll fuel the rise of antibiotic resistance. And there’s also some emerging thought that H. Pylori may actually be beneficial to some portion of the population since.
People carry this microbe, usually from childhood, and it can help train the immune system similarly to how we think of other microbes that are found in our microbiome. So trying to understand whom should be treated with antibiotics and when who is at risk of developing cancer down the line. That’s a very important question, and that’s some of what our work is focused on understanding.
00:05:46.238 –> 00:05:48.338 when you think about you know so
NOTE Confidence: 0.911280177916667
00:05:48.338 –> 00:05:50.209 much of the world’s population.
NOTE Confidence: 0.911280177916667
00:05:50.210 –> 00:05:53.186 Have this this bacteria you know
NOTE Confidence: 0.911280177916667
00:05:53.186 –> 00:05:55.680 a reasonable proportion of them.
NOTE Confidence: 0.911280177916667
00:05:55.680 –> 00:05:58.242 Get gastritis and ulcers and are
NOTE Confidence: 0.911280177916667
00:05:58.242 –> 00:06:00.620 typically treated with as you say,
NOTE Confidence: 0.911280177916667
00:06:00.620 –> 00:06:05.660 antibiotics and acid reducing medications.
NOTE Confidence: 0.911280177916667
00:06:05.660 –> 00:06:06.840 But there is this subset
NOTE Confidence: 0.911280177916667
00:06:06.840 –> 00:06:08.480 who go on to get cancer.
NOTE Confidence: 0.911280177916667
00:06:08.480 –> 00:06:11.336 So what do we know about that population
NOTE Confidence: 0.911280177916667
00:06:11.336 –> 00:06:14.897 and why it is that they are more
NOTE Confidence: 0.911280177916667
00:06:14.897 –> 00:06:16.741 susceptible to developing malignancy?
NOTE Confidence: 0.878027569565217
00:06:18.160 –> 00:06:20.472 Great question I. I think we don’t fully
NOTE Confidence: 0.878027569565217
00:06:20.472 –> 00:06:22.652 know and that’s something that a lot
NOTE Confidence: 0.878027569565217
00:06:22.652 –> 00:06:25.080 of research in the field is focused on.
NOTE Confidence: 0.878027569565217
00:06:25.080 –> 00:06:28.032 We have some indications as a field as to

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what might be increasing the risk among certain individuals and those can range from geography to diet to genetic background, but there are challenges also in making some of those associations. So certainly there are certain parts of the world in which the incidence of H. pylori associated gastric cancer is higher particular parts of South America? For example, there have been studies linking altitude to the risk of developing H. pylori associated gastric cancer, as well as the amount of salt in the diet as well as iron levels. So various environmental factors
are thought to increase risk and certainly genetic predisposition in some cases may play a role, although we don’t fully understand what those factors may be. A challenge there is that the microbe is found in such a large portion of the population. That it can be difficult to identify key factors that really predispose subset to the development of cancer risk and on the microbial side, which I haven’t really mentioned thus far. The Microbit itself has a very complex evolutionary history which in and of itself is super fascinating.
It’s thought to have evolved with humans since several thousands of years ago, over 60,000 years. It’s been with humans and thus the phylogeny, basically the evolutionary history. How this microbe has evolved. Can be used to trace migratory patterns of the human, the human race, and it’s really thought that the microbe evolves quite rapidly as well. Once it’s in a specific human host and thus it can be very difficult to assign specific microbial genetic patterns with cancer risk. Although there are,
I should note, some important proteins and genes that the microbe carries which do correlate very strongly with cancer risk in some individuals.

So, how do we kind of move that forward? I mean when we think about people who present to their doctor with stomach pain and ulcers and gastritis, they generally speaking will have an endoscopy and a small biopsy will be taken and sent to the lab and the lab will confirm that. Yes indeed they have \( H \) pylori. Is it, you know,
It is possible for that lab instead of just saying yes, you have H. Pylori to look at the particular features of that particular H. Pylori and say well, this particular brand of H. Pylori has an increased risk of you developing gastric cancer versus another brand of the same bacteria. Yes, I do think that that’s possible. It is possible to culture the microbes from human samples and assess at a genetic level if the microbe contains these.
These risk factors, and I think that’s one possible approach. Practically speaking, it may be a bit challenging, but as we have more advances with regards to genome sequencing and PCR based methods that can help us identify these factors quickly, I think that’s one approach. What is maybe more important as we move forward in the field is trying to identify early risk factors because maybe one thing I haven’t mentioned is that gastric cancer tends to present quite later in life, so with regards to H.
Pylori infection. As I mentioned, children usually are infected with the microbe when they’re very young. Typically we believe through household contacts with other family members who have the microbe. And it’s not until decades later that someone would present with gastric cancer. So this very very long lag phase from, you know, the infection in childhood to the development of gastric cancer. And once it’s diagnosed, it can be pretty late stage.
So that’s also challenging in terms of treatment. And so I think what’s needed are ways to assess much earlier. Whether or not someone is at risk and one way, as you mentioned, could be looking at the microbe and then others. Others may be trying to identify host factors that may be indicative of infections that may be heading down the road towards cancer, and that’s what I think a lot of research in this area is focused on, including the work of my own lab.
00:10:57.460 –> 00:10:59.572 is trying to identify effects that microbes may have on the host that could be translated into new diagnostic tests and indications of early cancer risk.

00:11:08.240 –> 00:11:10.688 So, so tell us more about that in terms of the work that’s going on in your lab, sure, so. What we’re trying to do is excuse me, is trying to understand how infection alters proteins in cells that are found in the stomach in a manner that may promote tumor growth.

00:11:28.350 –> 00:11:30.954 So how is it that the microbe interfaces with human proteins? interfaces with human proteins?

00:11:32.810 –> 00:11:34.182 How do they alter?
How do those interactions alter the proteins behavior in a manner that could promote the development of cancer, so to break it down a bit when you have an infection, your body's immune system will respond to try to clear the infection and that generally involves the recruitment of immune cells. And these immune cells will produce a lot of oxidants or free radicals. These are small molecules that are very reactive. They contain a lot of oxygen. They’re starved for electrons, so they react very readily with
00:12:03.639 –> 00:12:05.199 other molecules in your cells,

00:12:05.200 –> 00:12:06.754 and they can cause damage to cells.

00:12:06.760 –> 00:12:09.032 Because of this intrinsic chemical reactivity.

00:12:09.032 –> 00:12:10.168 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:10.170 –> 00:12:12.501 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:12.501 –> 00:12:14.664 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:14.664 –> 00:12:16.854 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:16.854 –> 00:12:18.616 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:18.616 –> 00:12:20.970 And one of the major classes of biomolecules that can get damaged by these oxidants or proteins and proteins are super important because they do a lot of chemistry in ourselves.

00:12:20.970 –> 00:12:22.370 They they generate energy,

00:12:22.370 –> 00:12:23.558 they help cells grow.

00:12:23.558 –> 00:12:24.746 They help them divide.

00:12:24.750 –> 00:12:26.490 They provide structure to cells.

00:12:26.490 –> 00:12:28.896 They help mediate interactions between cells,

00:12:28.900 –> 00:12:31.245 and these are all very important processes.
that if they become dysregulated,
so if they’re interrupted or inhibited,
messed with in some way,
teled to the development of cancer.
And so what we’re trying to understand
is when you have an infection and
all of these oxidants are produced
by the immune system,
some of those oxidants will damage proteins.
And when those proteins get damaged,
do they alter some of these processes
that could encourage cancer to form?
If that’s the case in our data points,
to indicates that that is.
Then can we identify you?
Some of these proteins as
diagnostic markers of cancer risk?

So if these proteins are getting damaged early on,
can we use them as indicators that cancer may be more likely down the line?

Yeah, so I was just about to say, I mean, that sounds like just fascinating work and I’d really like to dig a little deeper into that.

But first we have to take a short break for a medical minute.

So please stay tuned to learn more information about research and to detecting cancer causing proteins.
00:13:35.316 –> 00:13:37.899 with my guest doctor Stavroula Hatzios.
NOTE Confidence: 0.832647365238095
00:13:38.320 –> 00:13:40.685 Funding for Yale Cancer Answers
NOTE Confidence: 0.832647365238095
00:13:40.685 –> 00:13:43.050 comes from Smilow Cancer Hospital
NOTE Confidence: 0.832647365238095
00:13:43.130 –> 00:13:45.315 hosting a Smilow shares cancer
NOTE Confidence: 0.832647365238095
00:13:45.315 –> 00:13:47.940 survivors series June 22nd and 29th.
NOTE Confidence: 0.832647365238095
00:13:47.940 –> 00:13:50.806 Register at yalecancercenter.org or
NOTE Confidence: 0.832647365238095
00:13:50.806 –> 00:13:55.200 e-mail cancer answers at yale.edu.
NOTE Confidence: 0.832647365238095
00:13:55.200 –> 00:13:57.365 Over 230,000 Americans will be
NOTE Confidence: 0.832647365238095
00:13:57.365 –> 00:13:59.530 diagnosed with lung cancer this
NOTE Confidence: 0.832647365238095
00:13:59.604 –> 00:14:01.464 year and in Connecticut alone
NOTE Confidence: 0.832647365238095
00:14:01.464 –> 00:14:04.532 there will be over 2700 new cases.
NOTE Confidence: 0.832647365238095
00:14:04.532 –> 00:14:07.048 More than 85% of lung cancer
NOTE Confidence: 0.832647365238095
00:14:07.048 –> 00:14:09.250 diagnosis are related to smoking and
NOTE Confidence: 0.832647365238095
00:14:09.326 –> 00:14:11.894 quitting even after decades of use
NOTE Confidence: 0.832647365238095
00:14:11.894 –> 00:14:14.074 can significantly reduce your risk
NOTE Confidence: 0.832647365238095
00:14:14.074 –> 00:14:16.374 of developing lung cancer each day.
Patients with lung cancer are surviving thanks to increased access to advanced therapies and specialized care, new treatment options and surgical techniques are giving. Lung cancer survivors more hope than they have ever had before. Clinical trials are currently underway at federally designated Comprehensive cancer centers, such as the battle two trial at Yale Cancer Center and Smilow. Clinical trials are currently underway at federally designated Comprehensive cancer centers, such as the battle two trial at Yale Cancer Center and Smilow.
control non small cell lung cancer.

More information is available at yalecancercenter.org you’re listening to Connecticut Public Radio.

Welcome back to Yale Cancer Answers.

I’m doctor Anees Chagpar and I’m joined tonight by my guest doctor Stavroula Hatzios. We’re discussing some of her recent research and right before the break she was starting to tell us about H. Pylori. Now, for those of you who are just joining us, you know each pylori.
And we never really think about it necessarily as being associated with cancer. However, Savula tells us that it’s actually a leading cause of gastric cancer and the mechanism for that is something that she and her lab is working on discovering because not everybody who has H. Pylori gets gastric cancer.

Thank goodness, but some people do. And so stavroula right before the break you were telling us that one of the potential mechanisms of this, if I understood correctly, is that with this H pylori your infection with this H pylori your
immune system starts to kind of act on that infection. It kind of gets geared up as it would to any infection and starts manipulating some proteins and that those proteins might actually signal cancer. Is that right? Yes, that’s what we believe and what we’re investigating, and so we think that a lot of the inflammation that occurs during H. Pylori infection, which is accompanied by the production of oxidants. Those small molecules that are highly reactive, leads to changes in your cells, the proteins,
the DNA that helps nucleate cancer formation, and as you mentioned, we're focusing on the proteins. How do those change in a way that may promote tumor growth? Our lab is using some advanced chemical tools that allow us to identify specific proteins and human gastric cells that get damaged or modified by these oxidants produced during H. Pylori infection, so we can basically canvas the whole cell with these chemical tools and say what proteins are you getting.
modified by these oxidants and then independently look at these proteins using biochemistry and some biology. Other very interesting tools in our toolkit to ask. What happens when these proteins are modified that may promote tumor growth, and for that we use a number of different model systems, both in the lab and using a number of other different systems to look at tumor growth as a result of these modifications to the proteins. the long term goal here is to identify modified proteins that could be used to diagnose cancer.
risk much earlier in an infection. So you might imagine, if someone presents at a clinic with an H. Pylori infection, maybe there could be a biopsy taken where we look specifically for proteins that we’ve identified in the lab. Promote tumor growth and see if those have been changed in a way that aligns with that outcome. And if you can detect those small molecular changes very early in an infection that may help improve outcomes on the patient side.
so that that’s an interesting theory

stavroula, but one of the things that you mentioned before the break, which is true, is that there is this lag time right between when you get an infection when you have. Gastritis and when you may ultimately end up with gastric cancer, are there ways that we can manipulate these proteins or or reduce risk in some way? Once we identify these proteins, right? That is absolutely the goal. So not only could such proteins serve as indicators of cancer risk, but the nice thing about proteins, and particularly a lot of
the proteins that we study, is that a lot of them carry enzymatic function so there are enzymes. That means that they can perform different chemistry in the cell. They can perform chemical reactions and those are proteins that are really nicely targeted by small molecules by drugs. And thus if these proteins are involved in the infection response and down the line, increasing the risk of tumor growth can be targeted by drugs, then you also have the opportunity to develop new chemotherapeutics.
that could help actually treat cancers that result down the line from these infections. So not only are the proteins important as diagnostic indicators, but also carry the potential to be new drug targets for actually treating the cancer itself. Wouldn’t it be better if we were able to somehow manipulate these proteins to prevent cancer? Is that something that’s being looked at? Yes, I think we’re not quite there yet. We’re still at the early stages of trying to identify what these
proteins are and how they relate to the time course of cancer.

From the point of infection.

But yes, I think that there's very much a possibility to intervene at a very early stage.

If you do sort of a screen for such proteins at early points of infection,

perhaps in in an ideal scenario.

In a case of a childhood infection,

for example,

and if you see this sort of indication,

then intervene at that point.

with these drugs that I mentioned.
might be down the line to kind

of inhibit the activity that

could lead to tumor development.

You know you mentioned that

Actually carry this Helicobacter pylori in

our stomachs and for the majority of us.

Thank goodness we never have any problems.

There's a subset that get ulcers

and a subset even smaller that

that goes on to get cancer.

And I wonder whether.

The latter subset who get cancer is

a subset who actually get ulcers

versus they can get cancer without

having that intervening gastritis,
inflammation, kind of phase.

In other words, if I have an asymptomatic infection with H. Pylori and I just carry this, but it never really bothers me.

Am I at the same risk of getting gastric cancer? As a result of carrying that bug, as I would be if I not only carried H. Pylori but that H. Pylori went on to give me gastritis and ulcers and so on and so forth.

And then I get gastric cancer. Do you understand my question?

Yeah, I think so.
And I should note again that I’m not a clinician, so this is certainly not my area of expertise. But I will say my understanding is that the latter holds true, so those individuals who do develop ulcers, Gastritis are the subset that are at greater risk for developing cancer down the line and the model in the field is that each pylori induces infection in some host induces this the field is that each pylori induces infection in some host induces this chronic inflammation and in some hosts and some humans who have the. The microbe overtime that leads to the development of this inflammation.
In the tissue and that process is what seeds the development of the cancer decades down the line as well. So it does correlate strongly with the incidence of inflammation and people who have each pylori and that makes so much sense because we've seen that in other cancers as well, where it really is. This inflammation, the damage to the tissues. This idea that you get inflammation? You get fibrosis. You get free radicals. You get tissue which is not as well perfused.
That can lead then to cancers and so you know, presumably the tests that you’re developing to look at these infections might be something that very easily could be done at the time that somebody presents for a biopsy. Diagnosing the H. Pylori to begin with when they have symptoms of gastritis. The next question is, the case that you know these gastric cancers tend to emerge in an area of inflammation and we can kind of see in your research and that
00:23:26.481 –> 00:23:28.767 of others that the the pathway
00:23:28.767 –> 00:23:31.286 seems to be these free radicals.
00:23:31.286 –> 00:23:34.737 These small molecules and and damage to
00:23:34.737 –> 00:23:37.578 proteins and an inflammatory response,
00:23:37.580 –> 00:23:40.256 and so on and so forth.
00:23:40.260 –> 00:23:43.062 With your chemical toolbox where you’re
00:23:43.062 –> 00:23:45.670 looking at these altered proteins.
00:23:45.670 –> 00:23:48.046 Have you looked at that in
00:23:48.046 –> 00:23:49.630 other cancers as well,
00:23:49.630 –> 00:23:53.302 and isn’t necessarily the case that
00:23:53.302 –> 00:23:57.079 these are always related to microbes?
00:23:57.080 –> 00:24:00.223 Or is it possible that some inflammation
00:24:00.223 –> 00:24:03.068 may be due to other causes,
00:24:03.070 –> 00:24:05.090 but that the end pathway,
00:24:05.090 –> 00:24:07.346 the end result in terms of the small
00:24:07.346 –> 00:24:09.487
molecules and the tissue damage and the protein damage, et cetera?

With the inflammatory response is the same?

That's a great observation.

And yes, I think it is very likely to be the case that a lot of these same protein damage pathways are are shared or are common amongst other cancers.

We specifically, my lab specifically has not looked at other non infection associated cancers, but other labs have begun looking has not looked at other non infection associated cancers, but other labs have begun looking at this question and have certainly done it in other contexts as well, and I think that there will be an emerging picture of some proteins that get damaged.
By inflammation and oxidative stress and variety of contexts and that these may provide very important clues for the risk of cancer development, and I should also mention that one thing that we haven’t touched on is DNA damage, which is perhaps the more well known target of these oxidants that are generated during inflammation. And that’s something that is certainly very common across many different types of cancers resulting from infection or not. Oxidants can damage DNA. Directly, and that can lead to mutations and
instability of the genome that ultimately helps seed cancer formation as well.

Yeah, the problem with that though, is that as you mentioned, the nice thing about proteins is that potentially you can do something about it. So are people looking at, you know, trying to figure out how you can manipulate the system so that. You can kind of counteract DNA damage. My perception is that that’s a little bit more difficult.

I think you’re right. Yeah, I’m not familiar with specific work in that area, and I think it’s it’s a very important point that the benefit the
advantage to looking at proteins, which is still a very emerging area of research, is that you have the opportunity to intervene and actually do something about it. And they also have the added advantage of being both diagnostic indicators or providing some clues. What might be to come down the line, but also an opportunity to intervene through drugs? Small molecule approaches to help alter these outcomes? So I think for us, that’s why there’s such an exciting area of research,
particularly as they relate to microbes.

Yeah, you know, as you were talking about kind of these pathways and the way that H. Pylori works in terms of gastric cancer by you know, kind of getting the immune system to respond to it and then creating these.

It made me think about other infections that we know also cause cancer, but that are not bacterial so we know H. Pylori is a little bacterium that lives in our stomachs. But we also know that many other cancers are caused by viruses.

So thinking about hepatitis, for example,
the pathway seems to be very similar
In terms of you know, creating.
Inflammation and fibrosis and free radicals and so on and so forth.
Is there a difference in terms of how bacteria and viruses work
in terms of developing cancer?
And is it possible for your research to potentially look at virally mediated cancers as well?
Sure, I think that there are opportunities to apply similar approaches to viral infections that are associated with cancer.
And of course a lot of viruses have been connected to very important malignancies,
HPV, human papilloma virus, and cervical cancer. That’s perhaps one of the most well known connections, but there are several others, like hepatitis C, Epstein Barr virus, and the link between viruses and cancer has been explored for quite a long time and a lot more is known about how viruses can engage with the human cell to cause cancer. So some of the pathways may be similar, but we think that they may also be very distinct with regards to microbes, but at the same time, these approaches I think will be
very valuable for assessing viral infections and finding common pathways.

Doctor Stavroula Hatzios is an assistant professor of molecular, cellular and developmental biology and of chemistry at the Yale School of Medicine.

If you have questions, the address is canceranswers@yale.edu and past editions of the program are available in audio and written form at yalecancercenter.org.

We hope you’ll join us next week to learn more about the fight against cancer here on Connecticut Public Radio.

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